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The management of preterm labor.

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Preterm birth is the leading cause of neonatal mortality and a substantial portion of all birth-related short- and long-term morbidity. Spontaneous preterm labor is responsible for more than half of preterm births. Its management is the topic of this review. Although there are many maternal characteristics associated with preterm birth, the etiology in most cases is not clear, although, for the earliest cases, the role of intrauterine infection is assuming greater importance. Most efforts to prevent preterm labor have not proven to be effective, and equally frustrating, most efforts at arresting preterm labor once started have failed. The most important components of management, therefore, are aimed at preventing neonatal complications through the use of corticosteroids and antibiotics to prevent group B streptococcal neonatal sepsis, and avoiding traumatic deliveries. Delivery in a medical center with an experienced resuscitation team and the availability of a newborn intensive care unit will ensure the best possible neonatal outcomes. Obstetric practices for which there is little evidence of effectiveness in preventing or treating preterm labor include the following: bed rest, hydration, sedation, home uterine activity monitoring, oral terbutaline after successful intravenous tocolysis, and tocolysis without the concomitant use of corticosteroids.

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Inhibitory effect of terbinafine on reactive oxygen species (ROS) generation by *Candida albicans*.

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*Candida albicans*, the most important opportunistic fungal pathogen, is able to generate remarkable amounts of reactive oxygen species (ROS). Since ROS are highly cytotoxic, this mechanism may contribute to the pathogenicity of this yeast, including its invasiveness and the inflammatory response of the host. Terbinafine, a synthetic antifungal agent of the allylamine class, inhibits ergosterol biosynthesis at the level of squalene epoxidase. Furthermore, there is evidence that terbinafine at therapeutic concentrations can be considered a free radical scavenger *in vitro* and could exert an anti-inflammatory activity *in vivo*. In this study we investigated whether terbinafine affects the generation of ROS by *C. albicans*. Blastoconidia of the *C. albicans* strain 3153A were cultured in YEPG-medium and, subsequently, incubated with different doses of terbinafine (1, 10 and 100 microg ml<sup>-1</sup>) for 10 and 60 min, respectively. ROS generation was measured by lucigenin-enhanced chemiluminescence. Formation of ROS was considerably dependent on cell number. Chemiluminescence signals were measured at a concentration  $>$  or  $=$  1  $\times$  10<sup>6</sup> cells ml<sup>-1</sup>, with a maximum of 1  $\times$  10<sup>8</sup> cells ml<sup>-1</sup>. Already after 10 min of incubation with terbinafine, a dose-dependent significant inhibition of ROS generation was found ( $P < 0.05$ ), whereas after 60 min this effect was amplified. In conclusion, terbinafine reduced the ability of *C. albicans* to generate ROS. Besides the known effect on ergosterol biosynthesis, this mechanism may contribute to its antifungal action.

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